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Chronic pancreatitis with sinistral portal hypertension: A rare case report

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ABSTRACT

Due to its rarity as an etiology of massive upper gastrointestinal bleeding, left sided portal hypertension also known as “Sinistral portal hypertension” is often missed or diagnosed late. It often leads to isolated gastric varices which have a scope of different treatment modalities ranging from endoscopic, interventional radiological and surgical procedures. We report a case of 40-year-old male patient who was a case of chronic pancreatitis due to chronic alcoholism presented to us with history of massive hematemesis and black stools and was diagnosed as Left sided Portal Hypertension.

Keywords: Splenic vein thrombosis, Gastrointestinal bleeding, Splenectomy, Left sided portal hypertension, Chronic pancreatitis.

1. INTRODUCTION

Left sided portal hypertension, also called ‘sinistralportal hypertension’ is an infrequent cause of massive upper gastrointestinal bleeding. Splenic vein thrombosis secondary to pancreatic diseases is the most common etiology. Other rare causes include: hereditary thrombophilia, spontaneous splenic vein thrombosis, retroperitoneal fibrosis. Left sided portal hypertension must be suspected in cases presenting as massive hematemesis with normal hepatic function and splenomegaly (Seyfettin et al., 2007).

2. CASE REPORT

A 40-year-old male patient who was a diagnosed case of chronic pancreatitis due to chronic alcoholism presented to us with history of malena for 3 days and one episode of hematemesis a day before the admission. Patient did not have recent alcohol binge drinking. He had no history of episode of vomiting or retching preceding hematemesis. He did not report any bleeding from oral cavity or nasopharynx. He was not on any anticoagulants and was not on chronic NSAID use. On initial clinical examination, patient had pallor without any icterus, cyanosis or lymphadenopathy. There were no signs of liver failure such as ascites, altered sensorium.

On general examination, Pulse: 126/min, BP: 100/70 mmHg in supine position and 88mmHg systolic after 3 minutes of standing, JVP: raised at 11cm of water, there were no edema feet, on per abdomen examination revealed



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tender hepatomegaly and grade II splenomegaly. CVS examination revealed grade II ejection flow murmur in the pulmonary area. CNS examination: conscious, anxious. There was no constructional apraxia and flapping tremors.

Investigations revealed; Hb: 2.9 gm%, MCV: 66, HCT: 10.2, WBC: 5200/cumm³, Platelets: 1,70,000/ cumm³, Prothrombin time: 12.8, INR: 1.02, Urea: 42 mmol/liter, Creatinine: 0.7 mg/dl, SGOT: 55 units/L, SGPT: 14 units/L, Total bilirubin: 0.7 gm/dl, Albumin: 2.9 gm, Amylase: 30 units/L, lipase: 19 units/L, Hepatitis B and C serology: Negative.

Patient was hemodynamically stabilized with packed red cell transfusions. Injection octreotide infusion was given for 3 days with keeping presumptive possibility of variceal bleeding. After stabilization, upper GI endoscopy was done which revealed large dilated veins involving whole of proximal stomach. Fundus showed a very large bunch of isolated fundal varix with evidence of recent bleeding (Figure 1A, B, C). At the same time, patient was managed with endoscopic cyanoacrylate glue injection for obliteration of fundal varix.



Figure 1 Endoscopic images showing gastric varices.

Ultrasonography of abdomen was done which revealed enlarged liver with normal echotexture without any nodules, atrophied pancreas with calcification and moderate splenomegaly. Fibroscan of the liver was done which revealed median stiffness of 6kPa which denotes normal liver architecture. CECT abdomen was done which revealed chronic pancreatitis with splenomegaly with evidence of splenic vein thrombosis. Later on, during hospital stay, patient had an episode of hematemesis for which urgent interventional radiology opinion was taken and patient underwent Balloon occluded retrograde transvenous obliteration (BRTO) and embolization of gastric varices through glue insertion (Figure 2).

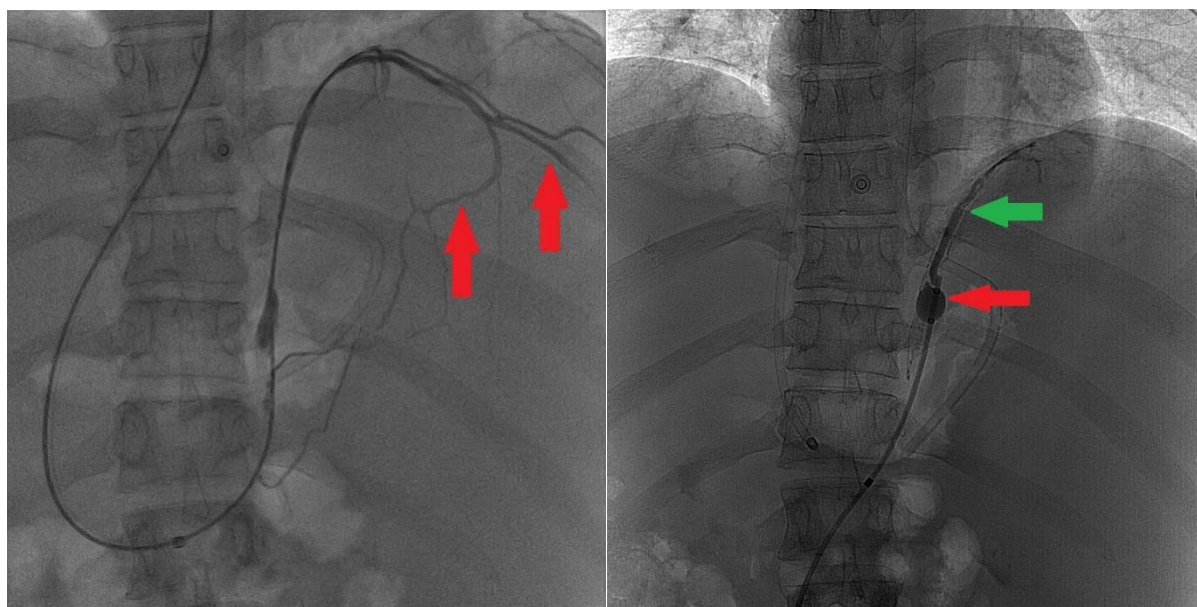


Figure 2 Left figure: Red arrows showing gastric varices by trans jugular approach. Right figure: As catheter could not be advanced in splenorenal shunt through trans jugular route due to less support, trans femoral approach was taken. Red arrow showing inflated balloon in splenorenal shunt and green arrow showing glue insertion in varices

Later on, during stay, patient again had an episode of massive hematemesis for which surgery opinion was taken for definitive management. Patient underwent splenic artery embolization followed by an open splenectomy (Figure 3).

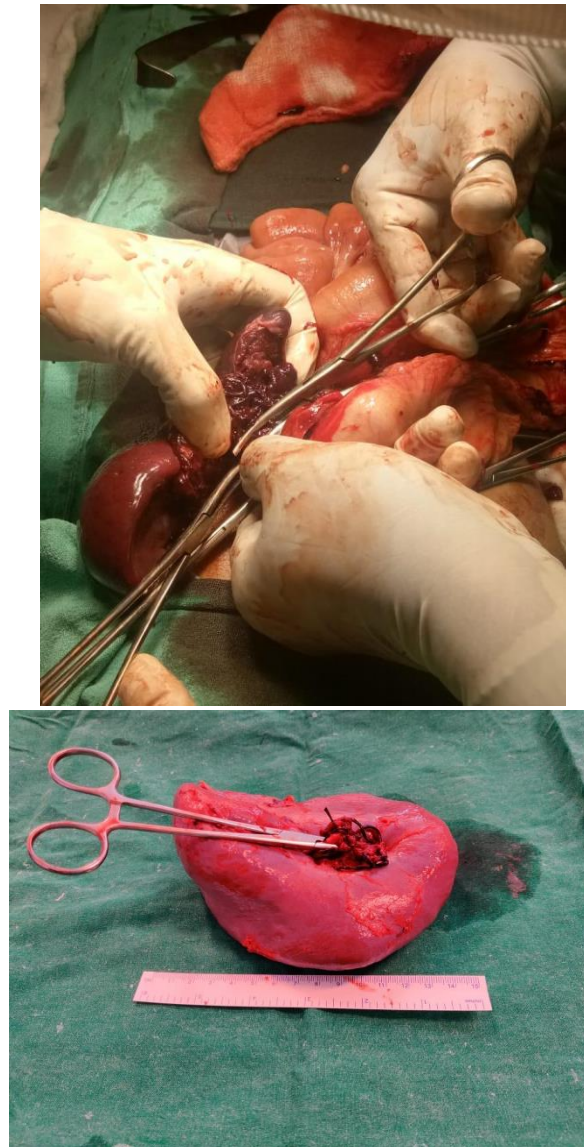


Figure 3 Images of splenectomy

Patient was discharged on post op day 15 with no post-operative complications and had been asked to come for checkup after a month in gastroenterology and surgery OPD.

3. DISCUSSION

Left sided portal hypertension is a different entity from usual cases of portal hypertension which has hepatic and/or extrahepatic portal vein involvement. In this entity, liver function is usually normal and extrahepatic portal vein is patent. It is usually due to obstruction of flow from splenic vein secondary to various causes. Splenic vein thrombosis is the frequent cause of left sided portal hypertension with other causes like pancreatic neoplasms and pancreatic pseudocyst. Splenic vein thrombosis can be secondary to many causes like chronic pancreatitis, hereditary thrombophilia, spontaneous splenic vein thrombosis (Ryan et al., 2004).

Sinistral portal hypertension cases are asymptomatic in most instances. They usually manifest as a gastric varix which is often an incidental finding and rarely it may rupture and present as an upper GI bleeding. Usually, upper GI bleed is secondary to cirrhotic portal hypertension. Thus, left sided portal hypertension is often missed and/or diagnosed late. Thus, it is quite useful to keep clinical suspicion for left sided portal hypertension as a cause of upper GI bleeding when there is normal liver function and splenomegaly (Kelin et al., 1981).

Asymptomatic cases of left sided portal hypertension are silent and often incidentally diagnosed on endoscopic examination. Asymptomatic cases are treated conservatively. There is no role of prophylactic splenectomy in asymptomatic patients (Shan-Hong et al., 2015). Case presenting with overt manifestations like upper GI bleeding usually warrants an intervention. Balloon tamponade, sclerotherapy, band ligation have been tried for control of variceal bleeding with minimal success. Endoscopic injection of cyanoacrylate glue for obliteration of gastric varices has been proved better for optimal hemostasis and lesser recurrence of bleeding (Shan-Hong et al., 2015). Balloon occluded retrograde transvenous obliteration (BRTO) is an interventional radiological procedure used in the management of gastric varices. The vast majority of gastric varices are associated with a spontaneous left-sided portosystemic shunt. BRTO involves closure of the portosystemic shunt with a balloon catheter, followed by injection of a sclerosant into the varix. The sclerosing agent results in thrombosis of the varices and draining portosystemic outflow, which marks an end of the procedure (Barbara et al., 2004).

In a case where endoscopic, interventional radiological modalities have failed, definitive treatment in terms of splenectomy is advised. Laparoscopic splenectomy has been advised over open splenectomy, but in our case, we had to opt for an open approach in view of unfavorable intraoperative findings (Garrett et al., 1990). Preoperative splenic artery embolization as a mean to reduce the venous pressure at the splenic hilum and to allow a safer and easier dissection of the splenic vessels represent an effective adjunct to splenectomy (Le-xin et al., 2012).

4. CONCLUSION

Left sided portal hypertension needs to be suspected in cases where patient presents with a triad of hematemesis, normal functioning liver and splenomegaly. Pancreatic disorders aid in the suspicion. With various modalities available to control the variceal bleeding, it has been tackled effectively, but definitive approach in terms of splenectomy should be put forth as early as possible in cases where bleeding has been refractory to other modalities.

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Conflict of interest

The authors declare that there are no conflicts of interest.

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Informed consent

Written and oral informed consent was obtained from the participant included in the study.

Data and materials availability

All data associated with this study are present in the paper.

REFERENCES AND NOTES

1. Evans GR, Yellin AE, Weaver FA, Stain SC. Sinistral (left-sided) portal hypertension. *Am Surg* 1990; 56:758–63
2. Heider TR, Azeem S, Galanko JA. The natural history of pancreatitis-induced splenic vein thrombosis. *Ann Surg* 2004; 239:876–882
3. Kohno K, Nakata K, Muro T, Furukawa R, Kusumoto Y, Munehisa T. Two cases of gastric varices due to pancreatic pseudocyst. *Gastroenterol Endosc* 1981; 23:1067–79.
4. Köklü S, Coban S, Yüksel O, Arhan M. Left-sided portal hypertension. *Dig Dis Sci* 2007; 52:1141–9.
5. Ryan BM, Stockbrugger RW, Ryan JM. A pathophysiologic, gastroenterologic, and radiologic approach to the management of gastric varices. *Gastroenterology* 2004; 126:1175–1189.
6. Tang SH, Zeng WZ, He QW, Qin JP, Wu XL, Wang T. Repeated pancreatitis-induced splenic vein thrombosis leads to intractable gastric variceal bleeding: a case report and review. *World J Clin Cases* 2015; 3:920–5.
7. Wang L, Liu GJ, Chen YX, Dong HP, Wang LX. Sinistral portal hypertension: clinical features and surgical treatment of chronic splenic vein occlusion. *Med Princ Pract* 2012; 21:20–23.